

ALTERED PULMONARY HEMODYNAMICS FOLLOWING EXPERIMENTAL
DECOMPRESSION SICKNESS

A. T. K. Cockett and Ray T. Kado

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From Department of Surgery/Urology, Harbor General Hospital,
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University of California (Los Angeles) School of Medicine.
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In previous reports^{1,2,3} we have described some of the patho-physiologic mechanisms which occur following experimental decompression. An increased pressure of 73.5 PSIG was maintained for 60 minutes; decompression was managed at the rate of seven PSIG - a lethal model for mongrel dogs.

An abrupt rise in pulmonary artery pressure following decompression has been reported earlier.⁴ Histopathologic studies by our group revealed the presence of pulmonary hemorrhage and edema of the pulmonary alveoli. Bone marrow emboli were found occasionally with gaseous and fatty emboli. These changes were found in the pulmonary arterioles.

Subsequent studies have suggested the development of a significant plasma deficit to account for the demise of the untreated animal.⁵ Replacement of plasma by dextran in dogs, or albumin in humans,⁶ have significantly reduced and virtually abolished the mortality rates.

A new pulmonary isotopic scanning technic developed in the dog by Taplin and his group⁷ provided the stimulus for this study. A method to assess the extent of pulmonary embolism is provided by scanning the pulmonary arteriolar tree prior to and immediately following decompression.

METHOD AND PROCEDURES

Sixteen dogs were used in the study.

1. Radioisotopic scanning

One hundred and twenty-five Micro-Curies of RISA $\bar{\text{I}}$ 131 is injected intravenously in the animals. The albumin particles previously iodinated with $\bar{\text{I}}$ 131 are of macro-aggregate size (40 micr). Work by Taplin and his

co-workers have demonstrated temporary blockage at the arteriolar-capillary junction. A nineteen hole focusing collimator is employed. Scanning is performed close to the skin - with the focal point $3\frac{1}{2}$ inches below the posterior rib cage.

2. Chamber procedure

All 16 mongrel dogs are splenectomized and allowed a 3-4 week recovery period. On the day of overcompression decompression 2 animals are selected prior baseline scans having been previously obtained.

The animals are anesthetized and overcompressed using a regimen we reported earlier.

3. Therapeutic regimen

a. Control - Following decompression one of the animals remains as the control while the second animal is treated with intravenous Dextran 350-500 ml. Post chamber lung scans are obtained $1\frac{1}{2}$ to 3 hrs. after decompression, and on a daily to biweekly basis for 1-3 months.

RESULTS

a. Untreated controls

1) Radioisotopic Lung Scan

All eight animals in the control group expired 1 to 6 hours following decompression. Radioisotopic lung scans revealed an increase in radioactivity in both lung fields. (fig. 1a, 1b)

2) Pathologic sections revealed pulmonary hemorrhage with marked edema. (fig. 2)

b. Animals Treated following Decompression

1) Baseline

Eight animals with prior baseline scans were evaluated following

decompression. Dextran fluid replacement had been employed as described earlier. Cold areas were observed in the upper left lobar lung fields.

2) A typical sequence of lung scans following decompression is illustrated in fig. 3a, b, c, d.

Radioisotopic lung scans revealed disappearance of the cold areas within 48 hours. (fig. 3c)

A second illustrative experiment (fig. 4a and 4b) also demonstrates a cold area developing several hours after decompression. Again resolution occurred after several days. These changes are no longer present 12 and 33 days after decompression (fig. 4c and 4d).

DISCUSSION

An abrupt rise in pulmonary arterial pressure following significant decompression may be attributed to the development of moderate pulmonary arteriolar-capillary blockade. Our previous studies would suggest that the pulmonary emboli responsible for the altered hemodynamic changes are fatty-gaseous particles. The pulmonary scans demarcate areas of emboli by marking the cold areas in the upper lobes. Baseline pre-chamber scans in these animals were free of such cold areas.

The pulmonary emboli apparently disappear in the dextran treated animals within 48 hours. The exact sequence of events occurring in the eight dogs still surviving two years since decompression needs to be elucidated. Further studies with biopsy of the involved sites is underway. Apparently, maintenance of an effective pulmonary arterial circulation - a mechanism accomplished by dextran replacement - is a major factor. A second probable mechanism is the development of collateral circulation in the involved areas. This apparently occurs within 48 hours since the cold areas are no longer

present after this period. Although conjectural, the development of A-V shunts initially in these areas is another distinct possibility.

The pulmonary scan is being further evaluated in our laboratory. We believe that semi-quantitation of the scan is necessary and possible. Only large macroscopic cold areas are detected at present. Subjecting the radioactive detectors (collimators) to a variety of filtering devices to screen out various energy levels is clearly indicated. The acquisition of such data which can then be evaluated by computer techniques is now underway in our laboratories.

Worth mentioning are the overall objectives of this long range study. We have described many of the hemodynamic and pathophysiological findings which occur following lethal decompression from depths in the 165-175 feet range. This model for decompression is probably applicable to depths beyond 250 feet. Whether such noteworthy survival rates can be obtained after more severe overcompression-decompression pressure changes needs to be determined. Accumulation of data, making possible the modification of decompression tables from depth (175-250 feet) on an emergency basis, continues to be our ultimate goal. Such tables may be helpful in exploring the continental shelf.

REFERENCES

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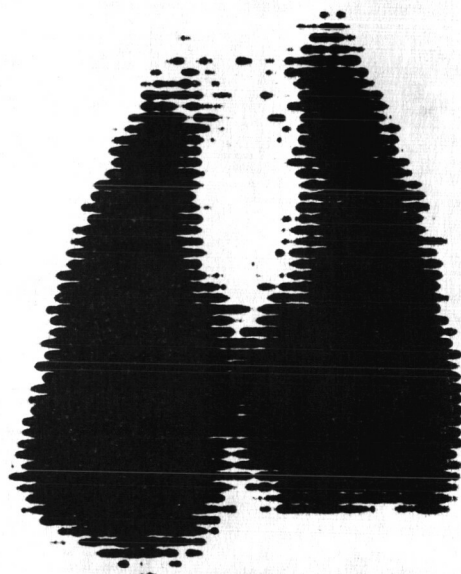
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Figure 1a

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64-2379

Figure 1b

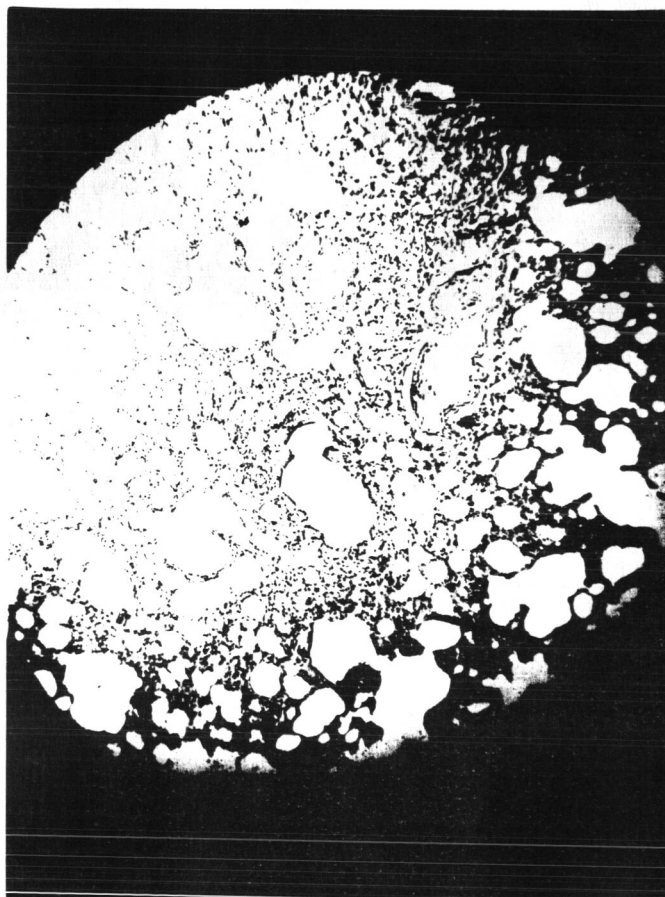
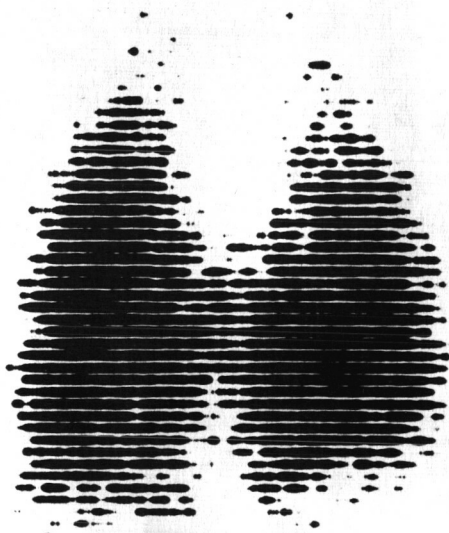


Figure 2

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Figure 3a

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Figure 3b

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Figure 3c

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64-1554

L

Figure 3d

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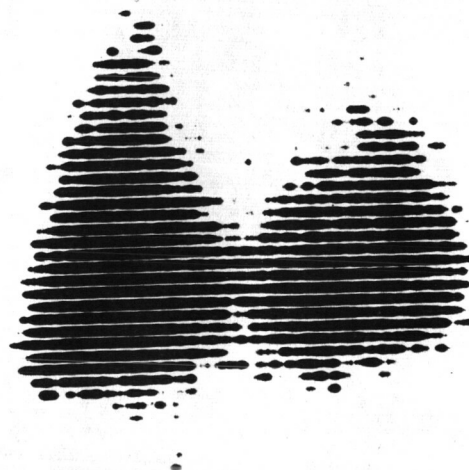


64-1561

L

Figure 4a

2



64-1561

L

Figure 4b

3



64-1561

L

Figure 4c

4



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L

Figure 4d

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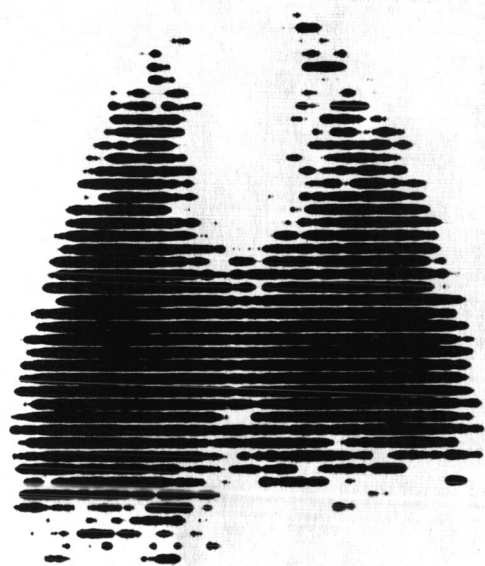
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Figure 1a

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Figure 1b

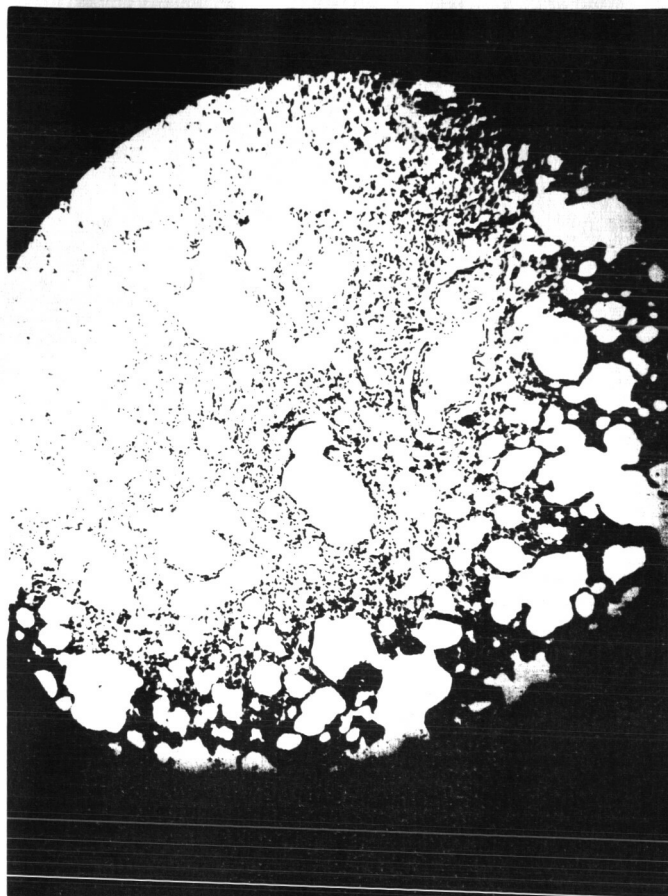
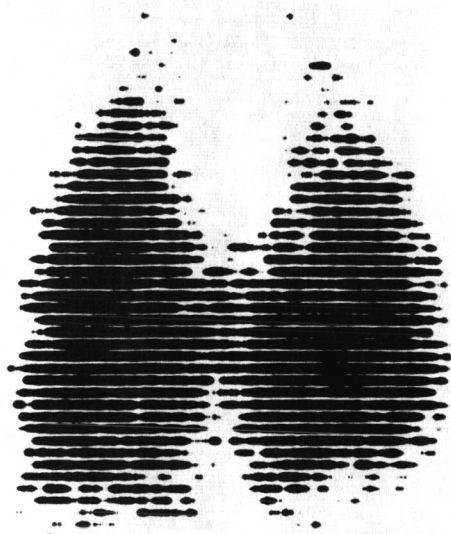


Figure 2

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64-1554

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Figure 3a

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64-1554

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Figure 3b

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Figure 3c

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64-1554

L

Figure 3d

1



64-1561

L

Figure 4a

2



64-1561

L

Figure 4b

3



64-1561

L

Figure 4c

4



64-1561

L

Figure 4d